retrograde pyelogram. The treatment of choice is nephrectomy with ligation of the fistulous tract.

A case is reported in which recovery followed operation.

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Meningovascular Myelitis in Early Syphilis

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ACUTE myelitis of syphilitic origin is a rare condition in the United States if the available case reports are a true indication of its frequency. For it to occur within six months following the primary infection, however, is still more unusual.[2, 3, 6] Many of the cases of acute myelitis reported during this period have been due either to the Jarisch-Herxheimer reaction following the first injections of arsphenamine or to a hemorrhagic myelitis from arsenical intoxication.[1, 4, 7]

When this tragic involvement occurs during therapy a differential diagnosis between these three possible causes is in many instances a difficult one to make. The neurological signs and symptoms of cord damage resulting from any one of them are more or less the same, regardless of the precipitating cause. If the patient recovers, credit is usually given to the method used in handling of the case and an etiological diagnosis is established on that basis. For the patient who dies, only examination of a cord section absolutely ascertains which process is the responsible one.

The following case report illustrates the problem involved under such circumstances:

CASE REPORT

The patient, a white female, was examined March 8, 1943. The skin on the entire body was covered with maculopapular eruptions of secondary syphilis and there were mucous patches in the mouth, as well as generalized lymphadenopathy. The patient complained of dull bifrontal headache. Results of blood tests by both the Kolmer and Kahn methods were four plus. Neoarsphenamine, 0.9 gm. intravenously, was given every fifth day for eight doses. Following the eighth injection, generalized arsenical dermatitis appeared. The patient was very ill, the skin of the entire body, including hair and nails, exfoliated, and there was a decrease in body weight of 40 pounds to a total of 115. After eight weeks during which vitamin therapy was the only medication, the patient began to improve and regained 20 pounds. A blood test on June 25 showed that the Kolmer reaction had been reduced to negative and the Kahn to three plus.

Around the first of September, however, the patient began complaining of intermittent deep aching of both lower extremities from the knees down, mostly when sitting. It was present day and night. Also, she complained of a progressive weakness of the knees and a fear of falling. There was incoordination of the legs in walking and she began staggering. The incoordination was worse on descending stairs but present also in ascending. There was "numbness" from the hips down, with decreased perception of touch and temperature. The patient was unable to void except with considerable effort and straining. Marked constipation was present.

A spinal fluid examination made at this time showed 83 cells per cu. mm.; a positive globulin; total protein was 0.107 mg. per 100 cc.; colloidal gold 1113331110, and Kolmer reaction two plus.

The patient had received no antiluetic treatment of any kind since April 20, the date the arsenical toxic skin symptoms first appeared, but in view of the positive luetic findings in the spinal fluid as well as the signs of progressive cord damage, specific treatment was again begun, this time starting with bismuth subsalicylate intramuscularly (0.13 gm.). After two months of bismuth therapy, no improvement was observed, so on November 11 1 gm. of tryparsamide was given intravenously. Nausea followed this injection but no skin reaction. Two grams more of tryparsamide was given on each of the following dates: November 18 and 26 and December 2. Neurological symptoms became more pronounced. The patient complained almost constantly of the sensation of a tight band or girdle squeezing the abdomen. The legs became progressively weaker, the patient fell to the floor and was too weak to arise without assistance.

Physical examination: The general physical examination revealed nothing unusual. The blood pressure, blood count and urinalysis were essentially normal.

*Neurological examination** revealed an uncertain gait, positive Romberg, moderate general wasting of the muscles and marked ataxia. Sensation to touch about the nose was diminished; that from T 10 was variable, with saddle anesthesia. Vibratory sensation was absent below the knees. Reflexes in the upper extremities were exaggerated bilaterally as were the patellar jerks, but the ankle jerks were absent. Plantar responses were equivocal. Examination of the cranial nerves gave essentially normal results.

More tryparsamide was given, followed by a gain in weight and general improvement. However, the bladder, bowel and leg paralysis became more complete. After eight weeks, cystitis, pyelitis and pyelonephritis developed rapidly, and death resulted from streptococcal septicemia and bronchopneumonia in February, 1944. A spinal fluid examination made a few days before death showed the Kolmer to be negative in all dilutions, and the Kahn two plus, the cell count 74 and the colloidal gold 0012332100.

Microscopic findings* in the spinal cord were reported as

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*The neurological examination and report and the pathological examination and report were made, respectively, by Dr. Helen Starbuck, San Francisco, and Dr. Melvin Friedman, University of California Medical School, San Francisco.

follows: Chronic leptomeningitis—fibrous thickening of pia mater and moderate lymphocytic infiltration.

Partial loss of nerve fibers in both dorsal and lateral funiculi, most pronounced in the dorsal columns in the cervical region and lateral pyramidal tracts in the lower thoracic region, suggesting a diffuse parenchymatous destruction within the cord.

Arteritis, syphilitic type, of the larger arteries.

Degeneration of a small percentage of nerve cells in inflamed areas, particularly the anterior horn cells.

Thick collars of lymphocytes around many small blood vessels in gray and white matter.

Intense hyperemia of tiny blood vessels in some inflamed areas.

Few small hemorrhages in gray matter, often but not always near a small blood vessel.

Absence of ring hemorrhages, perivascular necrosis, hydropic or fatty degeneration of endothelial cells, and diffuse uniform neuronal disease.

With the exception of the few small hemorrhages the evidence supports the diagnosis of syphilitic meningomyelitis, and this evidence is quite compatible with that diagnosis. In support of arsphenamine myelitis are the changes in the blood vessels and hemorrhages, none of which are specific or exactly typical, while against this complication is the last noted finding listing the more characteristic findings of arsenic reaction. In the opinion of the pathologist, therefore, the patient undoubtedly had syphilitic meningomyelitis as the principal lesion, and while a superimposed arsenical reaction of minor proportions cannot be entirely excluded, the evidence in its support is very feeble.

While the spinal fluid examination made at the onset of the cord symptoms indicated the presence of a definite luetic involvement of the nervous system and while the signs and symptoms of acute myelitis did not appear until nearly three months after the onset of the arsenical dermatitis, the fact that this patient had been so ill generally from the drug intoxication led to the belief that the arsenic intoxication had played a definite part in the nervous system damage. Bismuth therapy following the spinal fluid examination and later tryparsamide caused no improvement in the condition; in fact it seemed to worsen under antiluetic treatment. Also, the blood Wassermann had reversed from a 4 plus to a negative during the interval when no specific therapy was given and before the cord symptoms developed.

Altogether these observations strengthened the belief that the myelitis was of toxic origin and deterred the author from administering other specific therapy such as Swift-Ellis salvarsanized serum from a donor early in the disease when such therapy might have been of value. Also, the spinal fluid taken just before death showed a reversal of the Kolmer two negative, which again was interpreted as corroborating evidence of toxic etiology. The spinal cord sections, however, indicate that syphilis alone was responsible for the damage. Sections obtained at different levels of the cord showed the same inflammatory reaction. The meninges and vessels of the brain showed similar involvement. The microscopic diagnosis, therefore, was diffuse syphilitic meningovascular myeloencephalitis.[5, 8]

Nonne, a syphilographer of great experience, commenting on his observation of patients contracting syphilis late in life, said: "They are prone to develop early nervous system involvement and such processes in these patients are usually rapidly destructive."

SUMMARY

Acute arsenical dermatitis appeared in a patient being treated for secondary syphilis following the eighth injection of neoarsphenamine: The skin exfoliated, the patient became very ill, lost 40 pounds and for three months was hospitalized. During this period, although no further antiluetic treatment was given, the blood Wassermann became negative and the patient regained 20 pounds.

Symptoms of neuraxis involvement became manifest five months after the primary luetic infection. Spinal fluid findings were positive for syphilis, and, in spite of antiluetic therapy, paralysis ensued and the patient died of intercurrent infection.

Microscopic examination of sections of the cord showed the pathological process to be caused by syphilis and not by arsenical toxicity as was thought probable at the time of onset. This latter possibility prevented the use of more vigorous types of antiluetic therapy at a time when they might have been of value.

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